can play but a slightly important part in the general economy and physiology of the organism; or may it still be that the spleen can give heavily of its substance in famine and yet remain a valuable organ with divers and sundry useful though little known functions? Or does the spleen with specific purpose aid in the garnering and temporary storing of protein food materials in order that it may dole out sustenance when the body is hard put?

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GASTROSPASM: A CLINICAL AND ROENTGENOLOGICAL STUDY.[1]

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Even before the roentgen-ray era local gastrospasm, especially such that occurs at the entrance and exit of the stomach (cardio-

[1] Read in part by J. Roemer, M.D., at the annual meeting of the New Jersey State Medical Society, June 15, 1920.

and pylorospasm respectively), was clinically known. In 1881 Strumpell called attention to what he named esophagismus, describing the spindle-shaped dilatation of the esophagus, and Mikulicz demonstrated it by his esophagoscope. Spastic pylorospasm in the adult was first described by Kussmaul and in the infant by Still. Complete gastrospasm was mentioned by anatomists and physiologists (Home, Tiederman and Gmelin) in the early part of the nineteenth century. Local segmentation of the stomach was mentioned by Retzius, who found it at the autopsy of a man who died suddenly during the stage of digestion.

Williams described the human stomach to possess three natural physiological constrictions: (1) Near the cardia which corresponds to the incisura cardica (His); (2) in the middle of the stomach; (3) at the antrum. Banchi, Cunningham and Orr mentioned the physiological pseudo-hourglass constrictions. Buedinger found during a laparotomy a spastic hourglass opposite the seat of the ulcer. Simmonds found at autopsy an hour-glass stomach with no demonstrable lesion in any part of the stomach. Becky injected the human stomach, soon after death, with formol and often found contraction phenomena corresponding to an hourglass. Recently, Waldvogel[2] demonstrated by inflation of the stomach that gastrospasm is not an infrequent occurrence.

Whereas this historical introduction serves to show that the subject is not new, we must not lose sight of the fact that its clinical importance and the possibility of diagnosing it correctly are the achievements of the roentgen-ray study by means of the contrast meal. It must be emphasized that gastrospasm as a sign is demonstrated by the roentgen-ray, but its diagnostic significance can only be interpreted by a complete clinical study.

Gastrospasm was aptly divided by Holzknecht and Luger[3] into regional, circumscribed and total. Regional gastrospasm may occur in any part of the stomach; the most common locations are at the cardia and pylorus.

Cardiospasm. Although cardiospasm primarily belongs to the cardiac end of the esophagus, its relation to gastric diseases and its symptomatology are so purely gastric that brief mention of it must be made here. We understand by cardiospasm a spastic contraction of the cardiac end of the esophagus, of shorter or longer duration, sometimes lasting for weeks and even months and leading to dilatation above the contraction. From an etiological standpoint cardiospasm is in the great majority of cases purely functional. This is easily understood when we recall that the propulsion of food from the esophagus into the stomach is mostly controlled by the autonomic nervous system. That is why the esophagus can be entirely stripped of its musculature if the mylohyoid muscle remains and

[2] München. med. Wchnschr., 1911, No. 2.
[3] Mitteilungen auf dem Grenzgeb., 1913, p. 669.

the nerves be intact, without disturbing the passage of food from the esophagus to the stomach.

This is not the place to enter into a detailed discussion of the physiology of deglutition and of the epoch-making and conclusive experimental work of Kroenecker, Meltzer, Cannon, Krauss and others; but some mention must be made of the innervation of the lower end of the esophagus and cardia.

The chief nerve supply of the motor function of the lower end of the esophagus and cardia is derived from the vagus. The influence of the sympathetic nerve fibers on the lower end of the esophagus is today also conceded. The existence, however, of intrinsic nerves of the lower esophagus and cardia able to carry out the function independent of whether or not the chief nerve supply is intact was demonstrated by Cannon. These nerves are important when they are called upon to take up the expulsion of the food from the lower part of the esophagus to the stomach and when there is obstruction at the cardia on account of an organic disease of the vagus. We thus see the important role played by the vagus and its allied nerves in controlling the act of deglutition. The disturbance of deglutition due to organic affection of the vagus and its allied nerves, either centrally or peripherally, is well known, but functional diseases of the vagus may also bring about disturbances in the act of deglutition either by derangement of the vagus directly supplying the esophagus or by way of reflex. Reflex disturbances need not originate in the vagus; they may also originate in the sympathetic system. It is therefore reasonable to assume that cardiospasm is primarily an outcome of irritability of the vagus. The underlying factors bringing about such a state have been divided by Held and Gross,[4] into five groups.

In the first group are the patients whose vegetative nervous system is below par through inheritance. Almost throughout their lives they have some physical disturbance brought about by an unstable vagus or sympatheticus, and are also subject to cardiospasm. Although physically perfectly healthy, such people are found to have objective signs of vagotonia or sympathicotonia or the two mixed, and in the history we will hear that from childhood almost all organs supplied by the vegetative nervous system have at one time or another been subject to a functional disorder (enuresis nocturna, periodical vomiting, laryngospasm, urticaria, etc.).

To the second group belong the cases in which the vegetative nervous system becomes involved because of an inborn status asthenicus or status thymicolymphaticus. It is true that the status just named not rarely presents an inborn instability of the vegetative nervous system, but in a considerable number of cases we have found that the disturbance in the various organs brought on by

[4] Jour. Am. Med. Assn., June 22, 1916.

hyperirritability of the vagus and sympathicus is secondary. These individuals are not equal to the task of great responsibilities, either physically or mentally, and when overtaxed they need not necessarily acquire organic diseases, but they become subject to functional derangement of the vegetative nervous system. This may manifest itself in any of the organs supplied by the vegetative nervous system, and with equal frequency in the esophagus, causing cardiospasm. In this group belong the cases of gastroptosis, especially "long stomach" (langmagen), in which traction on the esophagus is the causative factor (Rovsing). Here also belong cases of cardiospasm resulting from sudden psychical trauma in individuals previously apparently healthy.

The third group contains the cases which show cardiospasm due to reflex irritation from other diseased organs (ulcus ventriculi, carcinoma of the fundus and lesser curvature, gall-stones, kidney-stones, diseases of the generative and genito-urinary organs, pancreatitis chronica or left-sided diaphragmatic pleurisy). Here, too, the underlying disturbed vegetative nervous system is a predisposing agent.

In the fourth group belong the cases of cardiospasm due to infections by toxic and metabolic agents (nicotin, lead, uremia, parasites, gout, chorea and lyssa).

In the final group belong the cases in which a local esophageal disease is the causative factor (fissure, erosions, scar-tissue formation and ulcus pepticum esophagi).

Symptomatology. It is important to know that the onset of cardiospasm is sudden and the first attacks may be of a short duration. Its transiency is best illustrated by two cases observed by us, one of a woman, who after a sudden shock by grief immediately developed cardiospasm which lasted several hours and recurred three weeks later. Another case is that of a man who presented clinical symptoms of cardiospasm which lasted three months. During fluoroscopic examination for half an hour the esophagus presented a characteristic sausage-shaped dilatation. Upon the administration of a Seidlitz powder, which was given merely as a suggestive measure, the spasm suddenly relaxed. These cases are cited to emphasize how frequently the affection is purely functional. In the majority of cases, however, once symptoms set in persistence is the rule. The severity of the symptoms varies, sometimes the patient can swallow solid food and no liquids, and at other times they can swallow liquids easier than solids. The severity of the symptoms depends greatly on the underlying causes. Often the patient will state that the first few morsels of food stop at the cardia and relief is experienced by taking more food; he even feels it going down into the stomach. When the condition persists, food stagnates in the esophagus, causing epigastric distress, substernal pressure, pain in the chest and belching and regurgitation of decomposed

food. There are often salivation and fetor *ex oris*. When extreme dilatation takes place the patient may have the false belief of feeling better, for he can partake of a larger meal. But if he takes a little more and reclines after it the food is regurgitated unchanged. Some patients vomit large quantities of food taken days before, simulating pyloric stenosis. This often leads to emaciation. In advanced cases the food is regurgitated in a state of fermentation and decomposition, showing the presence of lactic acid, butyric acid and sugar. In such cases due to these organic acids the patient has a continuous substernal burning. Pain as a symptom depends entirely upon whether the sensory nerves are co-affected or not. If so the pain is out of proportion to the disease and an attempt to swallow a little cold water causes severe pain in the region of the ensiform process, radiating to the soft palate, uvula, epiglottis, left side of the chest and sometimes both sides. Such pain is especially common if the spasm is due to a fissure at the cardia. A patient with cardiospasm observed by us was particularly troubled by pain as described above. Her blood showed a four-plus Wassermann and we hoped that antiluetic treatment might prove effective, which, however, was not the case. This made us doubt whether the existing lues was the underlying cause. Whereas the symptoms just described are typical of cardiospasm, very indefinite complaints such as feeling a fulness immediately after the smallest meal, belching and pressure in the epigastrium should lead us to examine carefully for a minor degree or cardiospasm. This is especially found in individuals of status asthenicus with a very long stomach in whom traction on the esophagus by the long stomach is supposed to cause spasm. To us it seems more plausible that the spasm is brought about by an undermined vegetative nervous system.

The physical examination should be directed not only toward establishing the disease, but also, if possible, to determine the cause; and therefore we should look for such clinical manifestations which indicate instability of the vegetative nervous system, *i. e.*, the vagotonic complex (Eppinger and Hess), such as dilatation of the pupils, slow pulse, slowing of the pulse when pressing on the eyeballs (Achner phenomenon), slowing of the pulse in bending forward, dry skin and pallor and spastic constipation. Not all symptoms must be present to complete the picture, and in reality some of the organs may show manifestations of sympathicotonia. Of course, it cannot be emphasized too strongly that the finding of symptoms of an unstable vegetative nervous system does not exclude organic disease. In fact it is to the credit of von Bergmann and his pupils, who demonstrated that such state predisposes to ulcer of the stomach and duodenum. It is surely of clinical importance to recognize the fact that in an organic disease like gall-stones or renal colic, if associated with cardiospasm, vagotonia plays quite a role as a predisposing causative factor.

Sounding. A soft stomach tube, not less than 10 mm. in width, should be used. In cases of spasm one has the sensation as if the tube were tightly held by the spastic esophagus. Sometimes sudden relaxation takes place and one feels the tube sliding down. The above-described sensation is more pronounced when a solid sound is introduced. The obstruction to the passage of the sound is not constant. Antispasmodics employed for a few days in the form of tincture belladonna, 10 minims, three times a day, or atropin, $\frac{1}{100}$ grain, two or three times a day, or papaverin, $\frac{1}{6}$ grain, three times a day for a few days, or benzyl-benzoate, 30 minims, three times a day, or making the patient drink some oil before the sound is introduced, may relieve the spasm.

Esophagoscopy. Most of the observers who use the esophagoscope with skill, such as Yankauer and Janeway, state that no definite information is obtained by this method. Both Rosenheim and Stark state that in certain cases hyperemia and in other cases ischemia were seen above the spasm, and also folds enclosing a narrow half-moon slit or even a pin-point opening above the spasm.

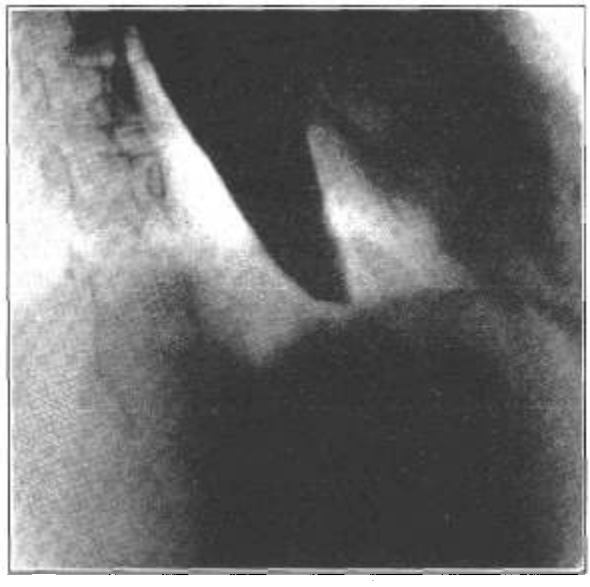

FIG. 1

Roentgen-ray Diagnosis of Cardiospasm. A most important radioscopic and roentgenographic sign is the visualized active peristalsis above the seat of the spasm. This is more marked in early and transient cases than in the advanced cases in which dilatation above the obstruction is very pronounced. The reason for that lies in the fact that the more the obstruction is due to spasm the more the

entire esophagus is in a state of hypertonus, and with its strong musculature tries to overcome the obstruction. In cases of obstruction at the cardia which result from idiopathic dilatation, where atony of the esophagus musculature is a primary factor, there is no peristalsis. Peristalsis of the esophagus is likewise absent in most of the cases of carcinoma at the cardia because of the associated loss of tone of the esophagus musculature. We differentiate two kinds of spasm: incomplete and complete. The former manifests itself fluoroscopically and roentgenographically as follows: The contrast substance is seen to hold at the point of obstruction and the esophagus above fills in an irregular spindle-shaped form exceeding its normal width two- or threefold. For a few moments nothing may be seen to pass through the obstruction. Repeated empty swallowing often overcomes the spasm. In the latter a uniform dilatation with visible contraction is seen above the obstruction. Through the obstructed area nothing may pass or a very thin streak of the contrast meal. If the condition persists for a very long time, dilatation may be so advanced as to permit the esophagus to hold from 200 to 300 cc of fluid. We can then see fluid in the dilated esophagus even without contrast substance. The esophagus looks sausage-shaped (Fig. 1), but shows peristalsis. The presence of peristalsis serves to differentiate cardiospasm from idiopathic dilatation or dilatation due to obstruction by cancer.

Rumination (Incomplete Cardiospasm). It seems of interest to us to record in this connection a case of rumination we had occasion to observe. While the patient swallowed the contrast meal which after a few morsels was seen to pass into the stomach with ease, the lower half of the esophagus would suddenly dilate and the patient would feel the food come back into the mouth. The patient would rechew it and swallow it again, then the food would suddenly drop into the stomach. This fluoroscopic study was an excellent demonstration of Meltzer's physiological studies as to the act of deglutition, *i. e.*, that the closure at the mouth of the esophagus during swallowing brings about the opening of the cardia (Figs. 2 and 3). Fig. 2 was taken immediately after the contrast substance was swallowed and Fig. 3 was taken one minute after the patient was asked to swallow empty. While fluoroscoping the patient during the act of deglutition we asked him to give a signal the instant he felt the food pass back into the mouth. The signal corresponded exactly to the time when the spasm in the cardia set in. This observation gave us a hint to advise the patient, instead of ruminating, to reswallow as soon as he felt the food back in the mouth or at the first sensation of distress in the epigastrium. Since then the patient reported repeatedly considerable improvement but not cured. Another illustration of how disturbance in the region of the mouth of the esophagus produces reflexly cardiospasm is demonstrated by a case of Zenker's diverticulum as shown in Figs.

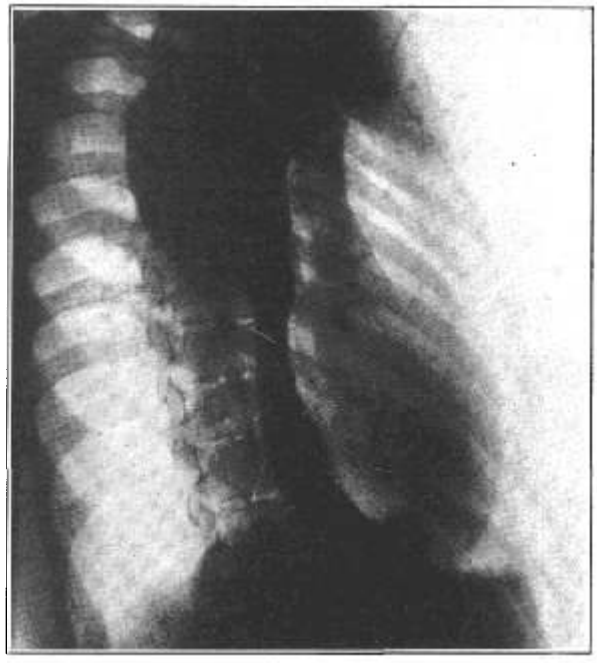

FIG. 2

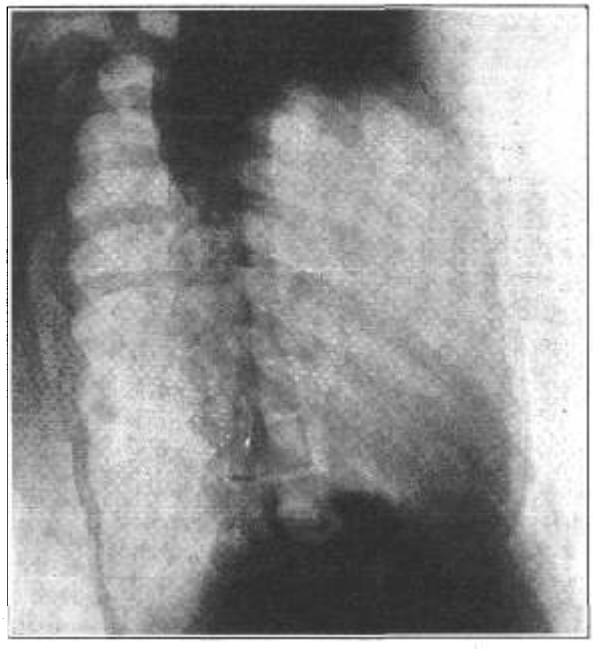

FIG. 3

4 and 5. (Fig. 4 shows the diverticulum and Fig. 5 the spasm.) Another case worth mentioning in which cardiospasm was caused

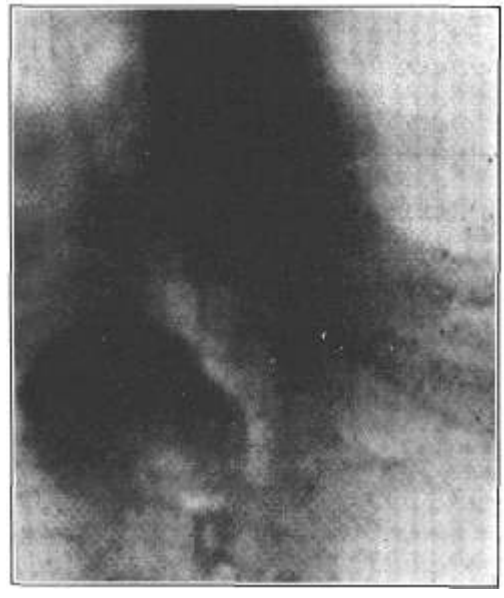

FIG 4

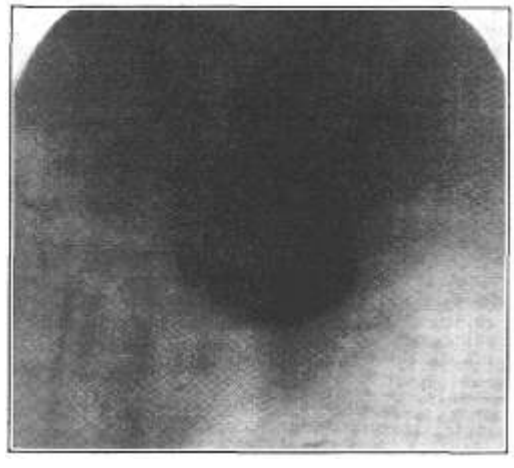

FIG. 5

by pressure in the upper part of the esophagus was due to an aortic aneurysm. It is interesting that these patients in whom the cardiospasm is brought about by disturbance in the upper part of the

esophagus, either as a result of disturbance of innervation (the case of rumination quoted above or anatomical defect, as in the case of Zenker's diverticulum, or if due to pressure from without, as in the case of aortic aneurysm) complain that the food stops in the throat or in the upper part of the esophagus.

Regional Spasm of the Stomach. Approaching the stomach proper the most frequent seat of regional spasm is that of the pylorus, so-called pylorospasm. Pylorospasm is a condition in which a part or the entire pylorus is in a spastic state for a shorter or longer duration. When a spasm persists for a long time it may even lead to hypertrophy and marked thickening of the pylorus with a resulting palpable mass simulating a tumor.

ETIOLOGY. Pylorospasm may be caused by intra- and extragastric disease (intrinsic and extrinsic—Carman). The intragastric cause is by far the less frequent, and if present is mostly the outcome of a prepyloric ulcer less frequently due to ulcer on the lesser curvature of the stomach and still less frequently in duodenal ulcer. We have seen it occur in gastro-enterostomized patients. Like Carman and Miller we saw it occasionally in carcinoma of the stomach.

The extragastric causes for spasm are numerous. According to our experience gall-bladder disease heads the list. Holzknecht and Luger were the first to call attention to the frequency of pylorospasm in gall-bladder disease, and they pointed out that the existence of a solitary stone in the gall-bladder is more prone to give rise to such spasms than multiple stones. Their observations were since then confirmed by Carman, Case and others. Our experience as demonstrated by Fig. 6 fully confirms their views. Other causes are chronic pancreatitis, chronic appendicitis, renal calculi, chronic lead-poisoning, morphine-poisoning, tabes dorsalis and chronic interstitial nephritis, particularly during the stages of suburemia and uremia.

SYMPTOMS. These, of course, vary, depending on the underlying cause. Where an intragastric cause like ulcer or cancer is responsible the well-known symptoms are present, which need no discussion. We consider it of sufficient importance to dwell more or less fully on the discussion of pylorospasm produced by gall-bladder disease, particularly gall-stones. Such individuals, independent of gall-stone colic, or even in the absence of any acute attack, have a long and persistent row of vague gastric symptoms. They complain almost continually of fulness in the upper quadrant of the abdomen. Just as soon as they begin to eat they feel that they have to stop because of pressure in the epigastric region, which is often relieved by spontaneous or forced belching. They often complain of pain of varying severity relieved by the application of heat. Such slight attacks generally occur during eating. Mental and physical exertions have a more deleterious effect than dietetic errors. The

tendency for these symptoms is to persist and eventually to lead to emaciation and pallor, and in extreme cases almost resembling carcinoma of the stomach. These symptoms, vague as they are, in the absence of definite physical findings, are often attributed to neurosis, and, in a sense, they are neurotic and functional in nature. These patients in the majority of cases have an unbalanced autonomic nervous system. By physical examination alone, no matter how painstaking it may be, it is very difficult to establish a diagnosis. Even the best palpation, including the Housman deep-sliding method of the pylorus, will seldom determine the existence of a spastic pylorus. In the pre-roentgen-ray era experts on palpation claimed to have succeeded in palpating a spastic pylorus.

FIG. 6

Experts, however, are the exception. The roentgen-ray, on the other hand, makes us visualize spasm with ease and offers an explanation for the symptoms.

The roentgen-ray findings are as follows: Fluoroscopically the food is seen to stop at the antrum, and sometimes even for several minutes no food at all is seen to pass, and what enters the pylorus appears in the form of a narrow canal or pivot-shaped. Palpation does not change the contour of the pylorus. The roentgen-ray plate (Fig. 7) shows the above-described phenomenon. The stomach proximal to the contracted pylorus shows dilatation and active peristalsis. It was already stated above that the clinical and roentgen-ray manifestations are such that to distinguish between these and carcinoma becomes very difficult. We may state that we, like others, had cases of persistent pylorospasm with anacidity.

These we submitted to operation with a doubtful diagnosis, but inclined to carcinoma. In 2 cases the pylorus was so hard and thick that the surgeon had to open the pylorus to convince himself that there was no tumor within. One of the cases was operated by Dr. Richard Lewisohn and the other by Dr. A. A. Berg. In cases in which the hypertrophied pylorus gives rise to a palpable mass and the contrast meal fails to fill the pylorus the differential diagnosis between spasm and cancer is almost impossible.

Regional spasm may occur in the fornix or tube of the stomach. In these cases it is brought about by pressure from without. We observed a patient in whom a large spleen made the upper part of the stomach from airbag to midpart of the tube appear like a narrow rigid canal with no peristalsis and a very small and deformed

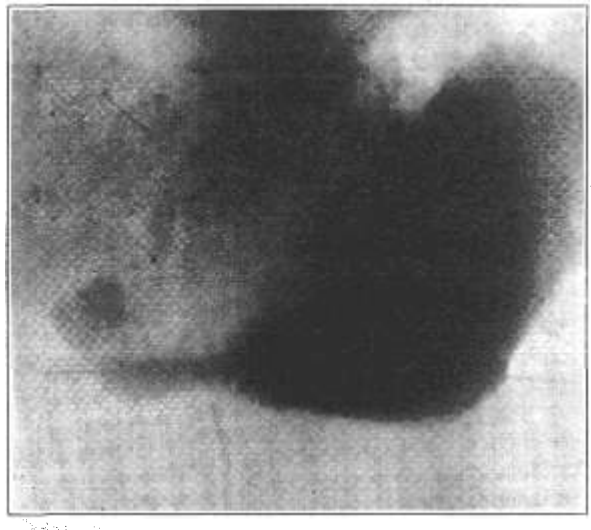

FIG. 7

air-bag. The palpable mass of the left side, which was the enlarged spleen, awakened the suspicion that the tumor might have been due to a malignant disease of the stomach. Inflation of the colon with air made the outline of the spleen to the palpating hand more definite A more extensive spasm of the stomach, taking in the tube and pylorus and making the cardiac end appear funnel-shaped, was seen by us in a woman with a large fibroid tumor of the uterus. A similar spasm of the fornix and part of the tube was seen by us in a case which at the laparotomy performed by Dr. William Spickers disclosed tuberculous peritonitis with tubercles scattered over the serosa of the stomach (Fig. 8).

Regional spasm of the tube or pars media, with dilatation of the cardiac end and the pylorus, is sometimes encountered. It is usually

of extragastric origin and only the clinical history can establish the cause.

A frequent seat of regional spasm is encountered in the stomach just below the incisura cardiaca. We do not refer to the standing

FIG. 8

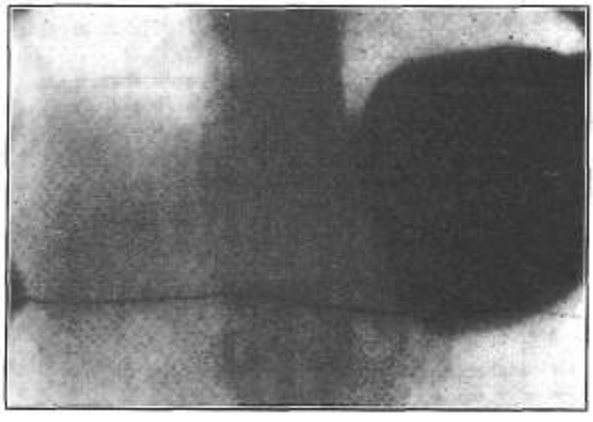

FIG. 9

contraction or the incisura on the greater curvature. This is to be discussed below. What we have reference to here is, as shown in Fig. 9, that the contrast substance is held at the point mentioned above. The fornix is seen dilated and sometimes from five to ten minutes either no food is seen to come down or only a thin streak

of contrast substance along the lesser curvature. In the former case the fornix presents a pear-shaped or triangular appearance (Fig. 10); in the latter case the narrowing below the dilatation bears resemblance to a cardiospasm with the dilated esophagus above it. We have therefore conceived the idea that the spasm is not only local, but that it involves the greater part of the tube. We were led to this observation accidentally, viz., through a patient who complained of severe choking sensation, pressure and fulness in the epigastrium after meals. The symptoms were at times so annoying that he would have to stop, when on the street, at any physician's office for the purpose of obtaining relief, for he feared lest he might choke. Nose and throat examination by specialists was pronounced

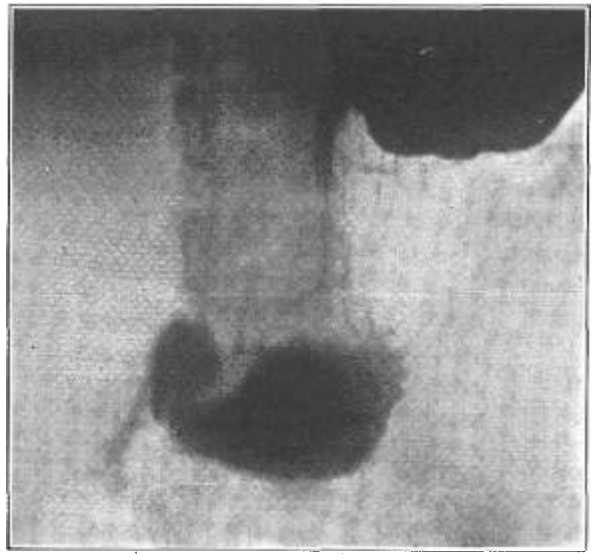

FIG. 10

negative. We studied his esophagus fluoroscopically with a contrast emulsion, as advocated by I. S. Hirsch, in order to exclude obstruction or a diverticulum of the esophagus. The esophagus was found to be normal, but as the emulsion entered the fornix it filled out that part and no particle of the contrast substance was seen to enter the tube. Massage of the fornix could not press the food down. Only after a few minutes a thin streak was seen to dribble down along the lesser curvature. It appeared to us as if we were dealing with an organic hourglass contraction. We then gave him the ordinary barium-buttermilk meal and the stomach filled completely. We had the patient return two days later after the administration of belladonna (according to the method of Carman) and repeated

the examination as before, and observed the same phenomena. A few days after the second examination we examined him again with the ordinary barium-buttermilk meal, when this phenomenon was entirely absent. When we then questioned the patient a little more in detail about his gastric symptoms most of his complaints, clinically, suggested gastric ulcer. The chemical analysis of the stomach contents showed marked hyperacidity. We placed the patient on a Sippy treatment, with gratifying results.

We were at once impressed by the thought whether the spasm below the fornix, just at the junction of the fornix with the tube, may not give rise to symptoms of spasm in the upper part of the esophagus similar to cardiospasm. We also reasoned that where there is an irritated area at a certain point in the stomach a meal that would tax that part of the stomach more than the buttermilk meal may demonstrate the functional disturbance brought about by such irritation.

We have since then adopted the procedure to start all our gastrointestinal roentgen-ray examinations with a few tablespoonfuls of the barium emulsion. We may state now definitely that normally this emulsion is seen to pass down the lesser curvature without stopping in the fornix. This emulsion fills out the fornix better than the barium-buttermilk mixture, and therefore outlines it when it would otherwise not be seen. Thus far our observations of such spasms have led us to believe that the cause for the same is intrinsic, and in the majority of cases is most likely due to an ulcer on the lesser curvature at the point where the ulcer exists. The spasm seems to us to be a protective phenomenon, analogous to the spasm in the sphincter pylori in cases of pyloric ulcer.

Another point of very frequent regional spasm is that of the sphincter pylori. This condition is often brought about reflexly by chronic appendicitis, colicomucosa and is also often functional. It has great significance in erosions and ulcer in the region of the sphincter pylori. Such erosions may give rise to periodical gastralgia, terminating in vomiting large quantities of fluid, but rarely solid food. Vomiting gives great relief; morphine, on the other hand, may make the condition worse. If the condition lasts a long time, dilatation and hypertrophy of the pylorus become so marked as to give rise to a palpable tumor. Schnitzler[5] reported a case in which a tumor the size of an orange was felt in the epigastrium. That patient was operated. At first the surgeon did not find any evidence of a tumor, but while palpating the pylorus the tumor reappeared. A similar case was observed by us. Schnitzler considers an erosion in the sphincter pylori analogous to an erosion in the sphincter ani, and advises pyloroplasty in such cases.

The fluoroscopic and roentgenographic appearance in such cases is most striking, as shown in Fig. 11. The pylorus appears as if

[5] Wien. med. Wchnschr., 1891.

suddenly cut off and is immensely dilated. Forcing the food out while fluoroscoping we see only a narrow stream of contrast substance which fills the first portion of the duodenum very thinly, giving it the appearance of a goose feather. Sometimes we may watch for several minutes without seeing any food going through the pylorus. If we fluoroscope and make plates of such cases an hour or two after the ingestion of the contrast meal very little is seen to have left the stomach. If the spasm is of extragastric origin it is usually very transient and there is only a very moderate delay in the emptying of the stomach, and, as a rule, no six-hour residue is present. If there be a large six-hour residue in such cases we usually find the rest of the contrast substance in the terminal ileum, which is considerably dilated. The cecum, as a rule, contains very

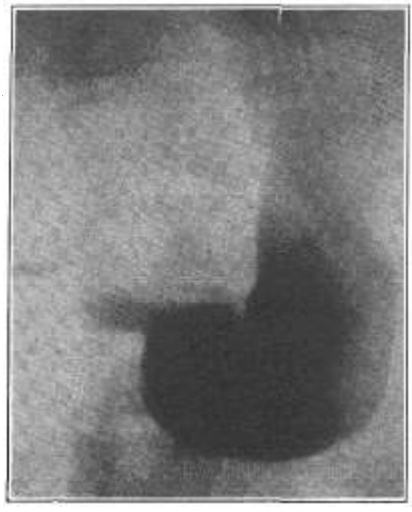

FIG. 11

little or no contrast substance at all. This seems to us to indicate the existence of a spasm in the sphincter of the ileocecal valve, and that is most likely the primary cause of the spasm in the sphincter pylori. The delay in the emptying of the stomach may not depend entirely on the persistence of the spasm in the sphincter pylori, but to a great extent upon the fact that the small intestines are filled, and therefore the so-called intestinal hunger (Pawlow) is not present.

When the spasm of the sphincter pylori is due to a local erosion the six-hour residue is large, the terminal ileum containing little contrast substance, or may be entirely empty, and the distribution through the colon may not be normal. The reason that the small intestine contains so little contrast substance, although the stomach

contains much, is explained by the fact that the food is so thoroughly liquefied in the stomach that it runs through the small intestines rapidly.

Incisura. The incisura or spastic hourglass has been made accessible of recognition by means of the roentgen-ray. It appears mostly on the greater curvature, a little below the incisura cardiaca. It may, however, appear in any other part of the greater curvature of the stomach. It may be transient or persistent. There is, usually, one incisura, but two or three may be present. The depth and width of the incisuræ vary from that of a small nick to a deep indentation reaching the lesser curvature and giving the stomach a bilocular appearance (Fig. 12). Where there are two or three incisuræ the

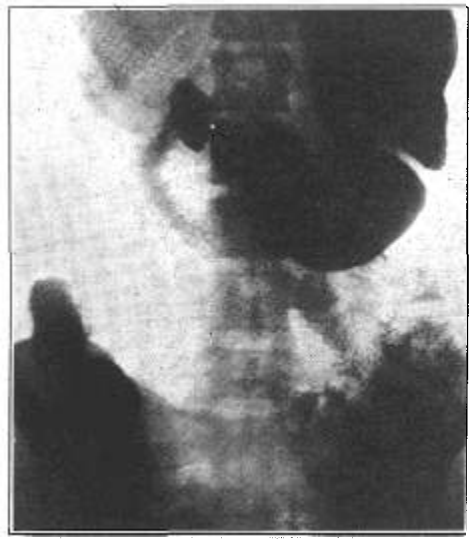

FIG. 12

stomach appears as if it were divided into several compartments. These incisuræ may be of extra- or intragastric origin. Such incisuræ are met with occasionally in cases of chronic appendicitis and gall-stones. Very rarely they appear spontaneously in such cases during fluoroscopic examination, but more commonly, as pointed out by both Case and Carman in this country and Borsany and Hurst abroad, such incisuræ are brought about when pressure is exerted over the diseased organ (appendix, gall-bladder and duodenum) while fluoroscoping. Even under such circumstances it is not a common occurrence. It has been our experience that when pressure on the diseased appendix or gall-bladder brings about such indentation it is very transient and lasts only as long as the pressure is continued. More commonly it is seen in neuropathic individuals,

especially when vagotonia is predominant. Also, pressure from without, such as a tumor in the left hypochondrium, a large spleen or even gas in the splenic flexure, may produce such spasm. The most important intragastric cause for spasm is an ulcer on the lesser curvature of the stomach opposite the incisura (Fig. 13). The spastic incisura, whether or extra- or intragastric origin, may be transient and disappear spontaneously while fluoroscoping or may persist during one examination and be present at the next examination. Deep and wide incisuræ, situated opposite an indurated or penetrated ulcer on the lesser curvature, are usually persistent and only disappear under deep narcosis. That is why the surgeon in the early days of roentgen-ray diagnosis contradicted the roentgen-ray

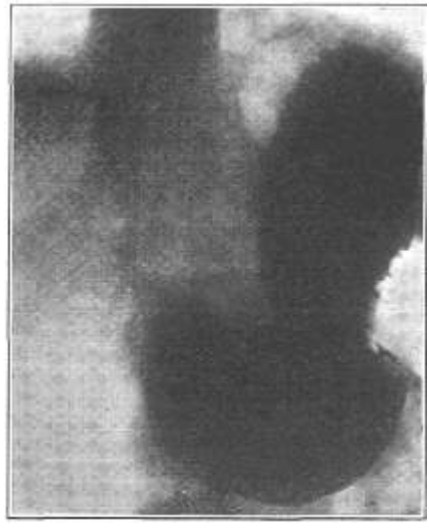

FIG. 13

findings of an hourglass. The differential diagnosis between spastic incisuræ of extragastric origin and that due to ulcer on the lesser curvature of the stomach is made by the administration of antispasmodics like atropin or tincture of belladonna, according to the method of Carman, or papaverin or atropapaverin, as advocated by Holzknecht and Skolitzer.[6] The disappearance of the incisuræ after antispasmodics, according to our opinion, does not exclude an ulcer on the lesser curvature. The persistence of the incisuræ, on the other hand, especially if a niche is seen on the lesser curvature, establishes the diagnosis of ulcer with absolute certainty.

A rare type of incisura occurs on the lesser curvature just below the air-bag, indicating the seat of an ulcer. A case with such incis-

[6] Wien. klin. Wchnschr., 1913, No. 26.

ura observed by us showed at operation no ulcer but a large solitary stone in the gall-bladder. Another rare form of incisura is mentioned by Faulhaber, from whose monograph Fig. 14 is taken. In this type the cardia is to the left and is accompanied by a narrow isthmus to the rest of the stomach which lies to the right. Primarily, Faulhaber correctly interpreted this as being due to an ulcer on the lesser curvature or to adhesions of the posterior wall of the stomach to the pancreas. Recently a number of publications appeared in foreign literature, notably among which are those of Rieder, Zehbe[7] and E. Schlesinger.[8] Rieder named this form of a stomach a cascade or waterfall stomach (Fig. 14). This form of a stomach presents a standing contraction or incisura on the greater

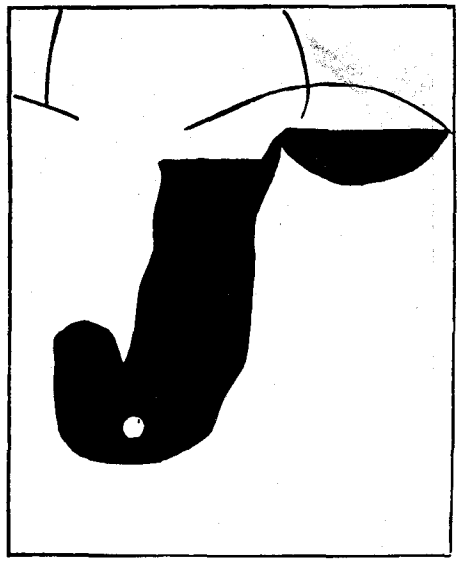

FIG. 14.

curvature just below the cardia, but instead of the corpus and pylorus running in the vertical axis the corpus and pylorus in the cascade stomach adopt the sandal or bull-horn shape. It seems justifiable to assume that such a stomach presents evidence of regional and partial gastrospasm combined. The regional spasm is demonstrated by the incisura and the partial by the narrow transversely situated tube and pylorus. That this condition is purely spastic is evidenced by the fact that it is not persistent and that it frequently disappears after the use of antispasmodics. Both intra- and extragastric disease play a role in the etiology of this condition.

[7] Fortsch. auf dem Grentz., Roentgen Strahlen, vol. xxv, p. 107.
[8] Ibid., vol. xxvii, p. 261.

It is a striking fact, which was observed by E. Schlesinger and confirmed by others, that duodenal ulcer frequently brings about such spasm. Fig. 15 shows a duodenal ulcer in a cascade stomach observed by us. Such condition can also be brought about by gas distention of the descending colon, a large spleen or tumors in the left hyperchondrium. In such a stomach the cardia or fornix is dilated, the air-bag is broad and presses against the left dome of the diaphragm with resulting clinical symptoms brought about from such pressure. This may also explain the frequent occurrence of left-sided pain in the duodenal ulcer. The organic hourglass of the

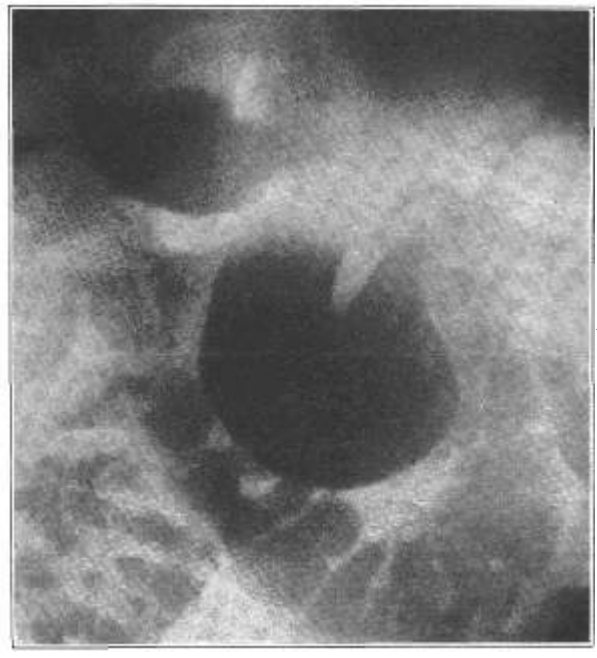

FIG. 15.

stomach due to ulcer, cancer and syphilis of the stomach will not be discussed here, as it does not belong to our subject. Before leaving the subject of incisuræ it seems in place to call attention to the fact that the phenomenon was much more frequently seen in the early days of gastro-intestinal roentgen-ray studies than now. This question has confronted us, and we thought that the reason for it may be in the difference of the consistency of the contrast meal. In the early days when a semisolid carbohydrate meal of Rieder was used the quality and consistency of the meal was of a nature to tax the perisystole in the fornix and upper part of the tube unless the stomach was very markedly hypotonic or atonic. A carbo-

hydrate meal remains longer in the fornix because salivary digestion continues there for a few minutes. The spastic incisura on the greater curvature has been considered as a protective phenomenon to prevent the food from irritating the ulcer-bearing area.[9] When a light fluid meal like the barium-buttermilk meal is given the meal is not sufficiently irritating to call upon such protective phenomena. A further proof that the lack of the filling of the fornix is responsible for the rarity of the incisura is furnished by the fact that when the fornix is made to fill by postural changes, such as having the patient lie on his back and obliquely (Levy-Dorn), an incisura may be brought out when it would not otherwise be seen. This cannot be due to local pressure from without because that position serves to evade pressure. We have therefore adopted the procedure to study all our cases in the Levy-Dorn position, with the barium emulsion as described above. If after such examination a conclusion is not reached the patient should be reëxamined with a Rieder meal. The reason we do not start the Rieder meal at first is because the fluid meal is better adapted for the study of the first portion of the duodenum and the appendix.

Total Gastrospasm. Total gastrospasm is the rarest of all forms of spasm and is in most cases of extragastric origin. Cases reported in the literature were due to chronic lead-poisoning, tabes dorsalis and morphinism. A case was seen by us in which the stomach looked like a narrow tube without any peristalsis and a gaping pylorus, and which we diagnosed as scirrhous cancer. This patient was operated by Dr. T. A. Dingman and no lesion was found in the stomach, but the appendix showed evidence of chronic inflammation. A case is mentioned in the literature which was due to gallstone disease. Carman reported a case of gastrospasm produced by a small cancer on the lesser curvature of the stomach. Gastrospasm is characterized by an extreme hypertonus. The stomach is considerably diminished in size. It is situated very high in the abdomen, most of it to the left of the median line. The air-bag is small, peristalsis is almost absent and the food is seen to pass continuously into the small intestine. It resembles almost entirely a scirrhous cancer. The resemblance of all forms of gastrospasm to filling defects due to cancer is so striking that rightfully does Carman make the statement that not only is the novice in danger of mistaking it for cancer, but the expert as well.

[9] Held and Gross: Am. Jour. Med. Sc., May, 1918.